Regular physical activity is considered to be part of a modern healthy lifestyle, and is widely recommended by both the medical community and clinical guidelines for the prevention and management of cardiovascular (and other) disorders (1). This general confidence in the health benefits of physical training is based on numerous epidemiological studies and other clinical evidence indicating that regular exercise may delay the development of atherosclerosis and reduce the incidence of coronary artery disease (1). Indeed, one meta-analysis has demonstrated that the relative risk of death from coronary artery disease for sedentary compared with active individuals was increased 1.9-fold (95% confidence interval 1.6–2.2) (2).

Nonetheless, not every sort of physical activity can be considered equally beneficial or even safe. Indeed, various reports demonstrate that vigorous physical training can transiently increase the risk of acute myocardial infarction and sudden cardiac death in susceptible individuals (1). Some cardiovascular conditions, such as atrial fibrillation, are more prevalent amongst habitual long distance runners (3) and – of course – an ‘athlete’s heart syndrome has been well described (4). More intensive types of activity such as jogging appear to carry a certain risk of unfavourable cardiovascular events (5). For example, in the Rhode Island study of the middle-aged men, despite the low overall incidence of death during jogging per se (that is, one death per year for every 7,620 joggers) this risk was still seven-fold higher when compared to individuals with more sedentary activities (5).

Which factors mediate the shift from beneficial effects of less intensive exercise regimes to potentially dangerous consequences of more intensive types of activity? Undoubtedly, these processes are complex and include numerous aspects, not all of which are entirely understood (6). For example, physical activity modulates the state of the coagulation system triggering either pro- or anti-thrombotic changes, depending upon the intensity and regularity of the training (7, 8). Also, the systemic and local (i.e. vascular) inflammatory status could perhaps represent one of many pathophysiological pathways (9–11). Indeed, an increased risk of developing cardiovascular disease, obesity, and diabetes with a sedentary lifestyle has been associated with a low-grade inflammatory status (12, 13). In a study of 1,383 asymptomatic individuals, for example, monocyte counts had the strongest independent relationship with overall cardiovascular risk, and other independent associations have been demonstrated between monocyte count and several cardiovascular risk factors such as blood pressure, body mass index, waist circumference, high-density lipoprotein (HDL) cholesterol and triglyceride levels (14).

However, even after high intensity bouts of exercise, monocyte levels have still been shown to completely normalise within 24 hours (19).

Also, different monocyte subsets appear to respond differentially to physical activity. For example, in the study by Simpson et al. (20), the proportion of CD14(+)/CD16(+) monocytes was 27% greater immediately after exercise, but was 49% less after just one hour post-exercise indicating fairly prominent dynamics of the ‘proinflammatory’ CD16+ monocytes. Moreover the profile of surface receptor was differentially affected on different monocyte subsets (20). Similarly, a moderate-intensity treadmill exercise bout predominantly increases CD16+ monocytes and reveals significant inter-subset differences with the observed response. Thus far, the reported exercise-related disturbances in monocyte numbers and function are short-term in nature and usually revert to baseline values within few hours (15, 16).

In contrast to acute exhaustive exercise, more prolonged physical training appears to have anti-inflammatory effects. Indeed, monocytes significantly decrease after a six-week course of moderate intensity cycling in overweight sedentary women, whereby monocyte counts were also significantly correlated with triglyceride levels, insulin sensitivity and body mass index (17). In another study, a 10-week training of sedentary individuals reduced serum tumour necrosis factor-α (TNF-α) level by 37% and decreased lipopolysaccharide-stimulated production of interleukins-6 and –1β, and TNF-α (18). Thus, the intensity of exercise training is undoubtedly important and exercise-induced monocytosis is more prolonged than compared to that seen after more intensive physical activity. However, even after high intensity bouts of exercise, monocyte levels have still been shown to completely normalise within 24 hours (19).

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changes in CD62L, CD11b, CXC2R, and HLA-DR expression (21). Indeed, physically active individuals have a lower percentage of CD14+CD16+ monocytes, lower unstimulated monocyte production of TNF-α and lower C-reactive protein levels compared to sedentary individuals (22). In the latter individuals, CD14+CD16+ monocyte percentage and stimulated TNF-α production was reduced after 12 weeks of exercise training (22).

In the January issue of *Thrombosis and Haemostasis*, Czepuch et al. (23) reported that a course of strenuous exercise in healthy sedentary individuals results in dramatic depression of monocyte migratory activity. Also, inhibition of monocyte migration towards vascular endothelial growth factor, transforming growth factor-β1 and monocyte chemoattractant protein-1 (MCP-1) was demonstrated. Of interest, these changes in monocyte function persist even at four weeks after completion of physical activity.

What are the implications of these observations? Short-term changes of monocyte migratory capacity could perhaps be expected on the basis of previous work (24) and are likely to reflect vascular adaptation and are likely to reflect vascular adaptation expected on the basis of previous work (24) and are likely to be cause or consequence of the training schema employed. Thus, the precise biological and potential clinical significance of the observations by Czepuch et al. (23) would need additional information on the same monocyte characteristics in physically active subjects as well as in patients with cardiovascular disease.

In conclusion, the benefits of regular moderate physical exercise definitely outweigh the potential risks in healthy subjects. Clearly, more research would need to be done in order that we have a better understanding of the physiological consequences of different types of physical activity.

### References


